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MINIREVIEW

Photosynthetic acclimation: State transitions and adjustment of photosystem stoichiometry – functional relationships between short-term and long-term light quality acclimation in plants

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In dense plant populations, individuals shade each other resulting in a low-light habitat that is enriched in far-red light. This light quality gradient decreases the efficiency of the photosynthetic light reaction as a result of imbalanced excitation of the two photosystems. Plants counteract such conditions by performing acclimation reactions. Two major mechanisms are known to assure efficient photosynthesis: state transitions, which act on a short-term timescale; and a long-term response, which enables the plant to re-adjust photosystem stoichiometry in favour of the rate-limiting photosystem. Both processes start with the perception of the imbalanced photosystem excitation via reduction/oxidation (redox) signals from the photosynthetic electron transport chain. Recent data in *Arabidopsis* indicate that initialization of the molecular processes in both cases involve the activity of the thylakoid membrane-associated kinase, STN7. Thus, redox-controlled phosphorylation events may not only adjust photosystem antenna structure but may also affect plastid, as well as nuclear, gene expression. Both state transitions and the long-term response have been described mainly in molecular terms, while the physiological relevance concerning plant survival and reproduction has been poorly investigated. Recent studies have shed more light on this topic. Here, we give an overview on the long-term response, its physiological effects, possible mechanisms and its relationship to state transitions as well as to nonphotochemical quenching, another important short-term mechanism that mediates high-light acclimation. Special emphasis is given to the functional roles and potential interactions between the different light acclimation strategies. A working model displays the various responses as an integrated molecular system that helps plants to acclimate to the changing light environment.

Introduction

In photosynthesis, light-driven redox chemistry of the electron transport chain and temperature-dependent enzymatic reactions of the Calvin–Benson cycle are tightly coupled. Any limitation in one of these two major parts of photosynthesis will have an immediate impact also on the other one. Therefore, the efficiency of the photosynthetic process is highly dependent on the ambient conditions in the respective habitat.

Abbreviations
Chl, chlorophyll; cyt b_6f, cytochrome b_6f; LHC, light-harvesting complex; LTR, long-term response; NPQ, nonphotochemical quenching; PQ, plastoquinone; PSI, photosystem I; PSII, photosystem II.

Changes in various abiotic factors, such as intensity and quality of the incident light, temperature, and nutrient and water availability, affect the photosynthetic yield. Thus, all photosynthetic organisms have evolved regulatory responses that acclimate their photosynthetic abilities to the actual environmental conditions [1–3].

Light is one of the most important environmental factors for a photosynthetic organism. As a result of various abiotic and biotic influences, light is highly variable in its intensity and its quality on both a short-term timescale (in the range of seconds to minutes) and a long-term timescale (in the range of hours, days and seasons). Important abiotic determinants include the geographical latitude of the ecosystem, the appearance of clouds and the leaf movement by wind. Biotic influences depend mainly on the individual position of a plant within an ecosystem, the density of the plant stand or, in case of algae, water depth and transparency [3–6]. In the last few years many advances in our molecular understanding of acclimation processes to fluctuations in light intensity have been made, for example, in the dissipation of excess high light as heat via nonphotochemical quenching (NPQ) through the action of the PsbS protein and the xanthophyll cycle (see the review by Horton *et al.* in this miniseries) and in the responses to photo-inhibition via the photosystem II (PSII) repair cycle [7,8]. Furthermore, much progress could be achieved in uncovering molecular mechanisms that participate in acclimation responses to low-light conditions, such as state transitions (see the review by Kargul & Barber in this miniseries). Field experiments with *Arabidopsis* mutants defective in these acclimation responses were used to test their importance in plants. The results clearly demonstrated that acclimation mechanisms, which so far had been mainly investigated under controlled laboratory conditions, have a beneficial effect on the seed production of *Arabidopsis* under field conditions [9]. Thus, acclimation responses contribute significantly to plant fitness and survival and provide a genuine evolutionary advantage.

All areas of high terrestrial photosynthetic productivity are typically characterized by plant communities of high density, such as forests or crop fields. In such dense plant stands direct sunlight is sensed only by the first leaf layers within the canopy or stand. In these layers, mainly high light intensities, which may harm the photosynthetic apparatus, must be counteracted, for example by NPQ. The incident light intensity in the leaf layers located below decreases exponentially as a function of the leaf area index, which describes the relation of the total leaf area of a plant (one side) to the covered ground area. This can range from 4 to 16 for different types of forests, and leaf area index values of 1–5 can be found in grasslands and in short-statured bushes [10]. Thus, sunlight is absorbed, reflected or scattered to a great extent in all plant communities. In all these habitats plants compete for photosynthetic active radiation, which leads to low-light conditions enriched with far-red wavelengths. Examples for such enrichment are shown in Fig. 1 (spectra in the open field versus spectra in sun flecks or shade). The effects of highlight acclimation responses is negligibly low and reactions to light quality gradients predominate. Quick changes in light quality (e.g. in sun flecks caused by leaf movement) are counteracted by state transitions in which the antenna structure of the photosystems is rearranged. However, most parts of dense plant stands typically exhibit light quality gradients of persisting nature. These are counteracted by a long-term response (LTR) that restores the photosynthetic energy balance by a re-adjustment of photosystem stoichiometry.

This high variability in photon flux density and spectral composition of incident light requires that photosynthetic acclimation responses are very dynamic and possess the capacity to respond to a very broad range of conditions (summarized in Fig. 1). We thus propose that the three different acclimation responses (NPQ, state transitions and LTR) complement one another. This implies that they are of different importance under the various illumination conditions mentioned above, leading to a rough functional hierarchy depending on the prevalent illumination conditions (condition 1, 2 and 3 in Fig. 1). The field experiments with *Arabidopsis* acclimation mutants mentioned above demonstrated a hierarchy in effectiveness in the order NPQ > state transitions > PSII core protein phosphorylation [9], which is consistent with our model for condition 1 (open field) (see Fig. 1).

At this point it should be noted that molecular analyses of responses to long-term far-red light illumination in recent years have focussed mainly on the role of photoreceptors (i.e. phytochromes), as in the case of the shade avoidance response, while the effects on photosynthetic acclimation responses have been investigated in less detail [11]. However, because both responses occur under the same environmental conditions and within the same time frame, at least some overlaps might occur. Early studies on photosynthetic acclimation uncovered a number of similarities in photosystem stoichiometry adjustment responses when tested by approaches using varying light quality, chlorophyll (Chl) *b*-deficient mutants or partial inhibition of photosynthesis by herbicides. This led to the

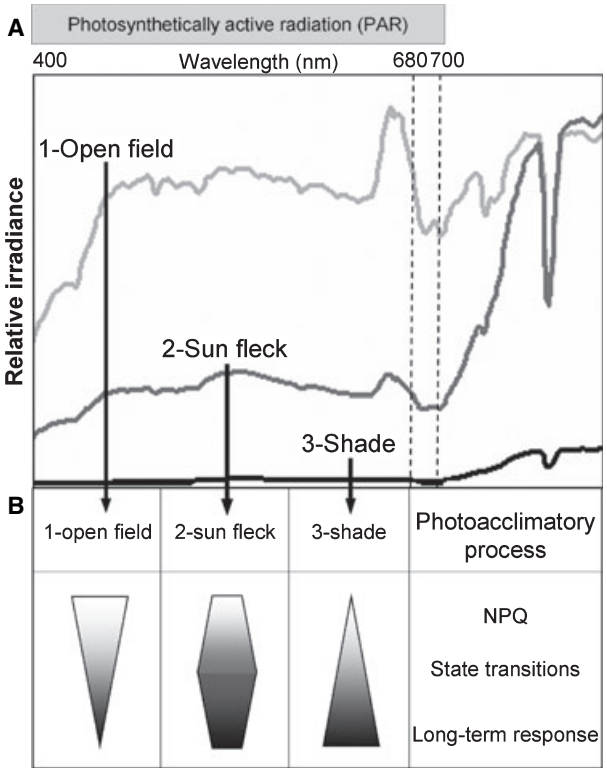

Fig. 1. Varying hierarchy of photosynthetic acclimation processes depending on the illumination condition. (A) Typical daylight light spectra measured under different conditions (recorded in May 2004 in the surroundings of Jena). The range of photosynthetically active radiation (400–700 nm) and the absorption maxima of PSII and PSI (680 and 700 nm) are marked along the top of the figure. Condition 1: daylight in the open field. Condition 2: a sun fleck within a dense population, exhibiting depletion in shorter wavelengths and enrichment of scattered far-red light. Condition 3: deep shade, exhibiting complete depletion of blue and red wavelengths usually used by plants for photosynthesis and enriched in far-red light. (B) Preferential action of acclimation responses under the different illumination conditions. Relative importance is indicated by the thickness of the symbol. Condition 1: NPQ is the major acclimation mechanism under full sunlight, where photosynthetic capacity is the limiting factor. State transitions serve for additional feedback de-excitation. Photosystem stoichiometry adjustment plays no, or only a minor, role. Condition 2: within a sun spot plants may transiently receive the full light spectrum with enriched far-red light. State transitions are most important to restore redox poise on a short-term timescale. Condition 3: under or in a plant canopy (permanent shade) the imbalanced excitation of photosystems is predominantly counteracted by adjustment of photosystem stochiometry as an LTR. NPQ plays no important role.

assumption that photoreceptors are not directly involved in photosynthetic acclimation responses [4,12]. This conclusion was confirmed by more recent studies, using phytochrome-deficient mutants, which clearly indicated that photosynthetic acclimation responses occur independently of the presence or absence of photoreceptors [13,14]. However, an interaction of regulatory networks at the molecular level cannot be excluded at the present stage of knowledge. Therefore, detailed and carefully designed physiological studies are necessary to unravel the relationship of photoreceptor-controlled and photosynthesis-controlled responses in addition to the relationships between the different photosynthetic acclimation responses.

Short-term response: state transition

The molecular events during the state transition process have been reviewed earlier in detail [15] (also see the review by Kargul & Barber in this miniseries). Here, we focus only on the basic steps that are important for understanding the relationship to the LTR. State transitions occur in the order of minutes and represent a mechanism in which excitation energy is redistributed between the photosystems by variation of their relative antennae cross-sections [16–18]. The variation is achieved by lateral movement of a part of the light-harvesting complex of PSII [light-harvesting complex II (LHCII)]. Upon reduction of the plastoquinone (PQ) pool, which transfers the electrons from PSII to the cytochrome b_6f (cyt b_6f) complex, a redox-sensitive kinase is activated which phosphorylates the mobile LHCII, resulting in its detachment from PSII and its attachment to photosystem I (PSI), in the so-called state 2. Under PQ oxidizing conditions the kinase is then inactivated and LHCII becomes dephosphorylated by the action of (constitutively active) phosphatases and is relocated to PSII (state 1). The mediation of the PQ redox signal towards the kinase is not yet understood; however, it involves the action of the PQ oxidation site at the cyt b_6f complex [19,20]. The LHCII kinase activity, however, is controlled by an additional regulation mechanism via the thioredoxin/ferredoxin system that inactivates it upon reduction. This effect occurs under saturating light conditions, when the stromal reduction state is very high [21]. The inactivation can be mimicked *in vitro*, for example by dithiothreitol treatment of thylakoid membrane preparations, strongly suggesting that the kinase activity is shut off by reduction of dithiol residues (see below) [22,23].

Recently, using mutant analyses in the alga *Chlamydomonas reinhardtii* and in the higher plant *Arabidopsis thaliana*, two orthologue thylakoid-associated kinases (TAKs), called STT7 and STN7 [24,25], were identified and found to be essential for state transitions. Mutants with defects in these kinases displayed no or much less LHCII phosphorylation and were not

able to perform state transitions, as demonstrated by the lack of characteristic differences in Chl fluorescence between State 1 and State 2. It is still not clear if these enzymes phosphorylate the LHCII directly; however, they provide an important tool for investigating the molecular regulation of state transitions that will facilitate future research in this field (see below). It is important to note that great differences in state transitions between *Chlamydomonas* and higher plants exist. In the unicellular alga, state transitions represent a very prominent acclimation process to light quality. Approximately 70–80% of the LHCII complex migrates during a state transition in *Chlamydomonas*, while in vascular plants only 15–20% of the LHCII complex moves [26]. Furthermore, in *Chlamydomonas*, State 1 and State 2 represent two different metabolic states. In State 1, linear electron flow is possible and equivalents (NADPH) are produced, while, in State 2, cyclic electron transport around PSI occurs which promotes ATP generation [16]. In higher plants, nothing similar has been reported so far. The evolutionary reason for these differences is not known. Possible explanations are that (a) plants might have to deal with more stable light environments, or that (b) unicellular organisms possess fewer energy resources than multicellular organisms and therefore have to respond more dynamically to survive adverse illumination conditions. Because of these differences one has to be careful with general conclusions on state transitions in plants and algae.

LTR: photosystem stoichiometry adjustment

The LTR is initiated whenever a photosynthetic organism is subjected to a stable light quality gradient for a longer time period. In the laboratory this can be easily investigated by growing plants for several days under artificial light sources that preferentially excite PSII or PSI (so-called PSII-light or PSI-light). Such light sources typically induce state transitions in the short term and therefore can be used to study both state transitions and the LTR. In contrast to the antenna movement during state transitions, the photosystem stoichiometry adjustment requires hours and days and redirects imbalances in excitation energy by changing the relative amounts of the two photosystems [4,27,28]. The principle difference between both processes is that state transitions represent a purely post-translational acclimation mechanism, while photosystem stoichiometry adjustment involves changes in the expression of photosystem genes and in the accumulation of Chl *a* and Chl *b* [29]. Despite the differences in the timescales of action, the LTR-like state transitions are triggered by the redox state of the PQ pool. Most species investigated exhibit enhanced expression of the PSI reaction centre genes *psaA* and *psaB* (encoding the P700 apoproteins) upon reduction of the PQ pool or a respective repression upon its oxidation. Examples that exhibit a respective opposite regulation of the PSII reaction centre gene *psbA* (encoding the D1 protein) have been also described [30]. Regardless of which regulation pattern is followed it finally leads to readjusted numbers of photosystems that support the redistribution of excitation energy and restore photosynthetic redox poise. Beside the altered photosystem gene expression, several other physiological and molecular parameters change during the LTR, including the Chl *a/b* ratio, steady-state Chl fluorescence, phosphorylation state of the LHC and photosystem core protein accumulation [13,31,32] (L Dietzel & T Pfannschmidt, unpublished results). At this point it should be noted that most of these parameters are also affected during long-term acclimation to changes in light intensity; however, it was demonstrated that *Arabidopsis* displays separate responses to low light and high light intensities [33]. In this review we focus on the LTR effects only in response to light qualities of low intensity. The LTR to light quality leads to an extensive restructuring of the thylakoid membrane system. Under PSI light the thylakoid membranes exhibit much stronger grana stacking and fewer stroma lamellae than under PSII light. This is accompanied by less accumulation of transitory starch in plastids from PSI-light-exposed plants when compared with PSII-light-treated plants [34,35] . Thus, it is not surprising that such a complex acclimation response involves the regulation of a great number of genes encoding products located in the plastids and also in the cytosol. Macroarray analyses revealed 286 redox-regulated genes covering all major functional groups, including photosynthesis, metabolism and signaling [13]. Inhibitor treatments indicate that at least 54 genes are regulated directly by the redox state of the photosynthetic electron transport chain. Thus, the data confirm that, as previously proposed, photosynthesis controls its own genes, not solely in the plastid but also in the nucleus [30]. In addition, it appears that the LTR also exerts control over processes downstream of primary photosynthesis, leading to a re-orientation in the metabolic network of plants by affecting, for example, carbohydate metabolism and the synthesis of nucleotides and amino acids [13] (K Bräutigam & T Pfannschmidt, unpublished results). The full extent to which the LTR controls gene expression and metabolism in higher plants is still poorly understood and will require systems biology approaches for an assessment.

Possible functional relationships of state transitions and LTR

Both short-term and long-term acclimation responses are controlled by the same redox signal in the thylakoid membrane and work in the same functional direction (which is to enhance the electron transport capacity of the rate-limiting photosystem); however, they act at different timescales. It has been hypothesized that the two responses represent functionally coupled processes [36,37], but it is still an open question of how these two mechanisms relate to each other. Do they represent two independent processes controlled by branched signaling cascades originating from the same signaling component (i.e. the PQ redox state), or are they two subsequent processes regulated by a single consecutive signaling pathway?

A recent study on the psychrophilic alga *Chlamydomonas raudensis* might give a first answer. Its natural environment is the Antarctic lake Bonney where blue–green low light (< 50 μE) predominates and the ciradian change of light quantity is diminished as a result of the long Antarctic day. These very stable light conditions make short-term reactions dispensable and, indeed, *C. raudensis* is not able to perform state transitions. However, *C. raudensis* retained the ability to perform long-term photosystem stoichiometry adjustment [38,39]. This indicates that the signal sensing mechanism was conserved during evolution in favour of the LTR, but that state transitions are not necessarily required for the LTR.

In *Arabidopsis*, studies on the *stn7* mutant uncovered an interesting connection between state transitions and the LTR. The Chl fluorescence parameter F_S/F_M [the ratio between steady state fluorescence and maximum fluorescence using the equation $(F_S/F_M = (F_t - F_o')/F_M)$] and the Chl *a/b* ratio were found to be useful for assessing the ability of plants to perform a proper LTR [13,31] because they exhibit characteristic differences between plants acclimated to PSI-light or PSII-light. The *stn7* mutant of *Arabidopsis* did not show such differences, indicating that this mutant lacks not only the state transition but also the LTR [40]. Thus, it appears that the STN7 kinase represents a common redox sensor and/or signal transducer for both responses. Consequently, the *stn7* mutant has been extensively used to uncover the physiological relevance of state transitions for higher plants. Under stable growth conditions with steady-state illumination the mutant exhibited no visible phenotype. However, in controlled growth chamber experiments with fluctuating intensities of white light or alternating illumination with PSI-light or PSII-light it showed retarded development in comparison to the wild-type plant [24,32]. In addition, in field experiments with field experiments with natural conditions the mutant produced $\sim 20\%$ less seed material than the wild-type plant [9]. All these observations confirm the general assumption that state transitions represent a process that counterbalances adverse effects on photosynthetic electron transport caused by frequent illumination changes under conditions of low light intensity.

The physiological relevance of the LTR for higher plants, by contrast, has, to date, been poorly investigated. An early study found an increase of $\sim 20\%$ in photosynthetic quantum yield (measured as oxygen evolution) of PSI-light or PSII-light acclimated pea leaves under the respective light sources [41]. However, state transitions were reported to change the excitation of PSI and PSII reaction centres but without any significant modification of the maximum quantum yield in CO_2 assimilation [42]. This suggests that gas exchange measurements are probably not sufficient to quantify the beneficial effect of light quality acclimation. Testing seed production has been shown to represent a useful approach that can be used for quantification of the beneficial effects of photosynthetic acclimation (e.g. NPQ) on plant fitness in *Arabidopsis* [43]. Using the *stn7* mutant as a tool, physiological experiments with PSI-light sources and PSII-light sources can be designed in which the role of the LTR for plant growth and reproduction can be determined. *stn7* mutants, grown in parallel with wild-type plants under PSI-light or PSII-light alternating every 2–3 days, produced $\sim 50\%$ fewer seeds than the wild-type plants. The same experiment, but with light shifts every 20 min, resulted in the same relationship of seed production between wild-type plants and the *stn7* mutant. However, the wild-type plant in the short-term light shifts produced $\sim 50\%$ fewer seeds than under long-term light shifts (R Wagner & T Pfannschmidt, unpublished results). Twenty-minute light shifts between PSI-light and PSII-light correspond to the time range of state transitions but prevent a LTR from being performed. Thus, this comparison indicates a clear beneficial effect of the LTR for seed production in *Arabidopsis*.

These first data suggest that light quality acclimation under low light conditions is very important for plants and that state transitions and the LTR both provide a significant benefit to a plant which is in a comparable order of magnitude. The two responses thus are coordinated in a temporally consecutive manner that covers a broad time range from very short-lasting to long-lasting excitation imbalances. Regulation of both via STN7 provides an elegant mechanism to couple

them in a physiological manner. Whether other components are involved in the regulation has to be elucidated in the future.

In this context, one open question arises from studies which demonstrated that not only the PQ redox state regulates the activation of the LHCII kinase. As mentioned above, there exists a second regulatory step in which the stromal ferredoxin/thioredoxin system inactivates the kinase under reducing conditions (e.g. under high light) [21]. Stt7 and STN7 possess two conserved Cys residues near their N-terminus. Mutations in these Cys residues abolish both LHCII phosphorylation and state transitions [15]. Because of a potential *trans*-membrane domain in this region it is likely that these residues are on the luminal side of the thylakoid membrane while the C-terminal kinase domain is on the stromal side [24] (Fig. 2). Inactivation of these kinases therefore would require a mechanism by which reducing equivalents from stromal thioredoxins are transduced to the Cys residues. Such a potential mediator might be HCF164, a thioredoxin-like protein that has been shown to mediate the reduction of luminal target proteins [44]. The electron transfer across the membrane might be performed by a protein called CcdA, which is a functional homolog of the bacterial DsbD protein that catalyses electron transfer from the cytoplasm to periplasmic target proteins [45]. It is difficult to reconcile such a thiol regulation with the PQ regulation in a physiological manner because, under reducing conditions, an activating signal from a reduced PQ pool and an inactivating signal from thioredoxin would act at the same time. A recent model proposed that thiol regulation can over-ride PQ regulation and plays the dominant role in LHCII kinase regulation under medium or strong white light [22]. A possible physiological explanation might be that by this mechanism the LHCII is locked to PSII and excess light energy can be directed to heat dissipation via NPQ, which protects PSI against photo-inhibition because it cannot not be repaired like PSII [32]. This coincides with the observation that major photosystem stoichiometry adjustments appear mainly in the low light range, suggesting that the LTR is shut off under higher light intensities and replaced by structural rearrangements for light protection [33].

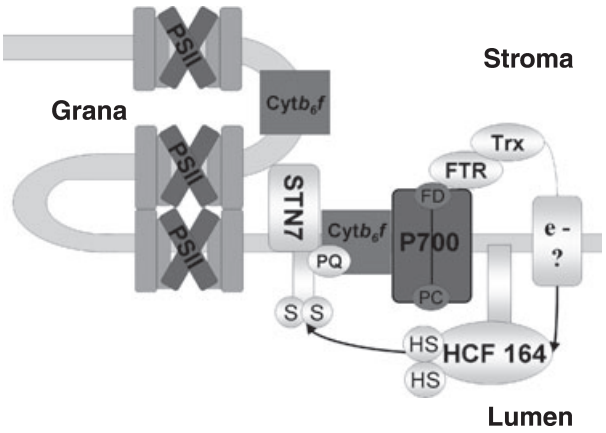

Fig. 2. Working model of STN7 regulation within the thylakoid membrane. The thylakoid membrane and integral protein complexes are drawn schematically. The abbreviations used are as defined in the text or at the end of this figure legend. STN7 kinase activity is regulated by the PQ redox state at the PQ oxidation site of the cyt b_6f complex and by the stromal redox state (ferredoxin/thioredoxin system) and sterical conformation of the thylakoid membrane. Reduction signals from PQ activate the kinase, whereas oxidation signals inactivate it. Reduction signals from PSI acceptors may over-ride this regulation and inactivate the kinase, even under PQ-reducing conditions. The information of the stromal redox state (NADPH) may be transduced to the lumen via a putative CcdA (question mark) protein that transfers reducing equivalents over the thylakoid membrane. The luminal thiol (SH) carrier, HCF164, represents a candidate that could regulate STN7 activity by reducing the N-terminal Cys residues (S) of the kinase. The stromal C-terminal kinase domain is probably too large to enter grana stacks. Therefore, grana destacking could be required for LHCII phosphorylation [15]. For further details see the text. FD, ferredoxin; FTR, ferredoxin/thioredoxin oxidoreductase; HS; PC, plastocyanin; S; Trx, thioredoxin.

Thus, besides the temporal co-ordination of state transitions and LTR in response to short-term and long-term light quality gradients, the STN7 kinase might also integrate light intensity signals. However, an increased number of physiological experiments (e.g. with the kinase mutants) are needed to unravel the regulatory coherences under these different illumination conditions.

Possible interactions of thylakoid kinases STN7 and STN8

Because photosynthetic redox control of state transitions and the LTR occurs in all photosynthetic eukaryotes studied so far, it seems reasonable to look more closely at the individual roles of thylakoid kinases [15]. Biochemical analyses identified a small family of three kinases, called TAKs. Antisense lines of these TAKs exhibited increased light sensitivity and were partially deficient in state transitions [46,47]. However, the precise biochemical function of the TAKs is still elusive. Besides phosphorylation of LHCII, phosphorylation of PSII core proteins (e.g. D1, D2, CP43 and PsbH) has also been found. Recent mutant analyses in *Arabidopsis* indicate that this process involves a homolog of the STN7 kinase, called STN8 [40,48]. It has been suggested that STN7 and STN8 might have slightly

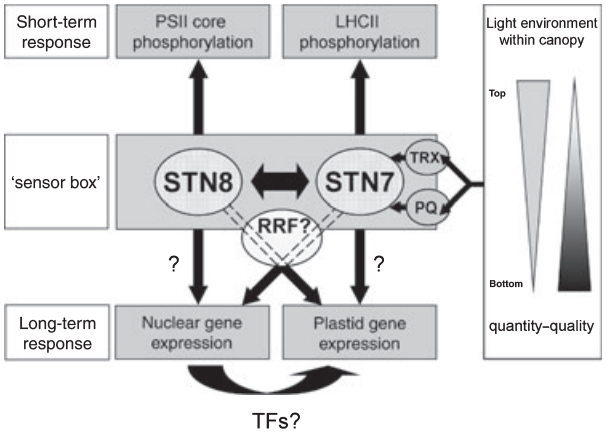

Fig. 3. Integration of state transitions and the LTR by kinase interaction. Both processes are regulated by information generated from photosynthetic electron transport. The redox signals of PQ and stromal PSI acceptors are integrated by combined sensing and action of STN7 and STN8, which form a so-called 'sensor box'. By this means various environmental illumination conditions (indicated by a box at the right margin) are integrated through their combined influence on the two redox systems. The light quantity gradient is illustrated by a grey triangle, whereas the changing light quality is depicted by a scale ranging from white (including sunlight) to black (far-red enriched scattered light). Redox control regulates not only LHCII phosphorylation but also PSII core phosphorylation (see the text). *stn7* mutants lost their ability to perform state transitions and the LTR. The photosynthetic redox signal could originate from the same sensor (probably STN7). During the LTR, expression of nuclear-encoded genes, as well as of plastid-encoded genes, is adjusted by a mutual interplay of STN7 and STN8 (see the text for details). The signal controlling nuclear and plastid genes could be mediated by (a) putative redox responsive factor(s) (RRF), which might be (an) additional substrate(s) of one or both of the two kinases. TFs, transcription factors; TRX, thioredoxin.

overlapping functions owing to the fact that some residual phosphorylation of LHCII in the *stn7* mutant and of PSII core proteins in the *stn8* mutant could be detected. These residual phosphorylations were not found in *stn7/stn8* double mutants [40], pointing to a possible interaction or cross-talk between both kinases in the wild-type plant (Fig. 3). A study using MS reported high specificities of STN7 and STN8 for different peptide substrates [48] but this does not exclude mutual interactions of these two kinases. Further support for this idea came from the observation that PSII core phosphorylation during the LTR changes in parallel with LHC phosphorylation, for example, PSII core phosphorylation decreased under PQ-oxidizing conditions (in PSI-light) or increased under PQ-reducing conditions (in PSII-light). This suggests that PSII core protein phosphorylation might be also redox-controlled (L Dietzel & T Pfannschmidt, unpublished results). Similar data were also obtained after only 3 h of illumination with PSI-light or PSII-light [32]. Another important hint on interactions of STN7 and STN8 came from the field experiments mentioned above. The *stn7* mutant exhibited slightly reduced seed production, whereas *stn8* behaved like the wild-type plant. By contrast, the double mutant *stn7/stn8* showed much more reduction in seed production, indicating synergistic effects when both kinases are lacking [9]. Additional interesting results were obtained by some gene-expression studies. The lack of LTR in the *stn7* mutant suggests that the control of nuclear photosynthesis genes might be disturbed. s*tn7* mutant plants, however, displayed no significant changes in transcript profiles when compared with wild-type plants, suggesting that the STN7 kinase activity, as such, has no direct effects on the expression of nuclear photosynthesis genes [32,40]. Surprisingly, such differences were observed in the *stn8* mutant but were masked in the *stn7/stn8* double mutant [40]. This suggests an indirect impact of STN7 on nuclear gene expression by acting on STN8. Alternatively, expression analyses with *stn7* mutants have to be carried out under very specific physiological or temporal conditions to uncover a clear impact on expression profiles. For instance, a recent study reported that STN7 might be involved in the circadian regulation of nuclear photosynthesis genes [49].

The present data point to a complex regulatory network that controls photosynthetic acclimation to illumination changes. We therefore propose that the two kinases STN7 and STN8 (and their counteracting phosphatases as well as unknown interaction partners) may build up a thylakoid 'sensor box', which integrates environmental influences on photosynthetic electron flow (caused by variations in light intensity and/or quality) by sensing and processing redox signals from the PQ–cyt b_6f complex and/or the ferredoxin/thioredoxin system (Fig. 3). The interplay between the kinases and their substrates integrates these varying redox signals and initiates appropriate molecular acclimation responses in the short-term and long-term range. This requires the existence of redox responsive factors that may transduce the redox signals to the level of gene expression in plastids and nucleus. The existence of a high number of unknown eukaryotic transcription factors within plastids has been proposed [50,51]. It will be a challenging task to understand the mechanistic interactions within this regulatory network that are responsible for controlling state transitions and the LTR. Molecular and genetic approaches are required to uncover as-yet-unknown components. Functional models can then be developed

by including appropriate physiological approaches combined with systems biology techniques.

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